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CASE REPORT

Topical Glucocorticoid-Induced Cushing's Syndrome in a Patient with Erythrodermic Psoriasis: A Case Report

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Background: Topical glucocorticoids are widely used in psoriasis treatment but may lead to systemic adverse effects, particularly with prolonged use. While pediatric cases are well-documented, adult-onset iatrogenic Cushing's syndrome from topical corticosteroids remains under-recognized.

Case Presentation: A 31-year-old woman with a 10-year history of psoriasis vulgaris self-administered high-potency clobetasol propionate ointment (monthly cumulative dose escalated from 30 g to 100 g over 22 months) under no physician supervision. She presented with erythrodermic psoriasis and Cushingoid features (moon facies, violaceous striae, pitting oedema). Laboratory tests confirmed suppressed cortisol (<1.5 nmol/L) and ACTH (2.86 pg/mL). Management included gradual withdrawal of topical steroids, initiation of oral methylprednisolone (8 mg daily) for HPA axis support, and transition to low-potency desonide. Four-month follow-up showed normalization of cortisol levels (167.1 nmol/L at 8:00 AM).

Conclusion: This case highlights the systemic risks of unsupervised, escalating use of high-potency topical corticosteroids in adults with impaired skin barrier. It underscores the need for adherence to dosing guidelines, patient education, and routine endocrine monitoring.

Keywords: Cushing's syndrome, dermatologic diseases, erythroderma, glucocorticoids, psoriasis

Introduction

Psoriasis is a chronic, relapsing inflammatory dermatosis with a global prevalence of approximately 2–3%. Clinically characterized by erythematous plaques, scaling, and dysregulated immune activation, it is associated with significant quality of life impairment and mental health comorbidities, necessitating timely and effective therapeutic intervention for symptom control and relapse prevention.[1] Topical glucocorticoids remain the first-line therapy for mild to moderate psoriasis due to their potent anti-inflammatory and immunosuppressive properties.[2] However, prolonged administration or high-potency topical glucocorticoids may lead to cutaneous adverse effects, including skin atrophy and telangiectasia. Although relatively rare, systemic absorption can occur, particularly after prolonged use, potentially resulting in complications such as iatrogenic Cushing's syndrome.

Iatrogenic Cushing's syndrome most commonly results from chronic systemic glucocorticoid exposure and is characterized by central adiposity, hypertension, hyperglycemia, and suppression of the hypothalamic-pituitary-adrenal (HPA) axis.[3] Emerging evidence suggests that topical glucocorticoids can achieve systemic concentrations through transdermal absorption, especially when applied in large quantities on inflamed skin or as high-potency formulations.[4] Such systemic exposure has been documented more frequently in pediatric populations and in applications in thin or sensitive skin regions, such as face or perineum. Documented cases in adults with intact skin on the trunk or extremities

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remain relatively uncommon. Although rare, systemic absorption leading to iatrogenic Cushing's syndrome has been estimated to occur in <1% of adults using super-potent topical steroids long-term. Key predisposing factors for iatrogenic Cushing's syndrome in psoriasis patients encompass the use of occlusive dressings, extensive skin surface area coverage, pre-existing barrier disruption, high potency and prolonged use of topical corticosteroids, and individual metabolic variations in steroid metabolism.[5] Interindividual variability in cutaneous absorption, glucocorticoid metabolism, and treatment adherence may contribute to susceptibility.

This report presents a case of iatrogenic Cushing's syndrome developing in an adult patient with psoriasis following prolonged application of high-potency topical glucocorticoids. The case highlights the potential for serious systemic effects from topical therapies and the necessity of physician vigilance in monitoring and managing patients on long-term corticosteroid regimens. This report uniquely documents rapid progression to erythrodermic psoriasis triggered by unsupervised dose escalation in an adult, contributing to the sparse adult literature on systemic corticosteroid toxicity from topical therapy.

Clinical Data

A 31-year-old woman presented with a two-week history of diffuse erythematous desquamation and bilateral lower limb edema. The patient had a documented 10-year history of psoriasis vulgaris, previously managed with oral acitretin and intermittent methotrexate therapy combined with topical corticosteroids, which had provided adequate disease control. In April 2019, topical therapy with clobetasol propionate ointment (10 g/vial, 20–30 g per application) was initiated, leading to rapid remission of cutaneous erythema. However, symptom recurrence occurred upon discontinuation of treatment, prompting repeated use. By December 2019, thinning of abdominal skin was noted after which the patient began applying petroleum jelly in conjunction with a corticosteroid. Recurrent disease flare-ups resulted in progressive increase in clobetasol administration, with monthly consumption rising to 30–40 g by January 2020 and further escalating to 70–100 g by October 2020. This intensive regimen persisted through February 2021. This study was conducted with approval from the Ethics Committee of Wuhan Asia General Hospital Affiliated to Wuhan University of Science and Technology to publish the case details, written informed consent to participate and for publication of images was obtained from the participant.

Patient self-purchased clobetasol propionate ointment from community pharmacies without physician prescription between April 2019 and February 2021. Approximately two weeks following discontinuation of the topical glucocorticoids the patient developed generalized erythema with scaling, paresthesia (tingling sensations) over the chest and abdomen, and bilateral lower extremity edema. A weight gain of approximately 10 kg was reported during the course of glucocorticoid therapy. Medical history revealed no pregnancies, surgical intervention, or concurrent systemic medications during this period. Systemic physical examination demonstrated no other significant abnormalities.

A dermatological examination revealed diffuse erythema affecting the entire body, with mild superficial scaling in certain areas, including abdominal distension and moon facies. Multiple wide, parallel, atrophic striae of dark red to violaceous hue were observed on the chest, abdomen, and lateral thighs, oriented both horizontally and vertically. Bilateral lower limb edema was also present (see Figure 1).

Laboratory investigations demonstrated significantly suppressed adrenal function. Plasma cortisol level measured 2.01 nmol/L at 00:00 AM, < 1.50 nmol/L at 04:00 AM, and 1.61 nmol/L at 08:00 AM (reference range: 64–536 nmol/L). Adrenocorticotropic hormone (ACTH) concentration was 2.86 pg/mL (AM reference range: 6.0–40 pg/mL), indicating suppression of the HPA axis. Blood glucose was within normal limits at 3.9 mmol/L (reference range: 3.9–6.1 mmol/L). Hypoproteinemia was evidenced by a serum albumin level of 25.8 g/L (reference range: 40–55 g/L), and a total protein level of 47.4 g/L (reference range: 65–85 g/L).

Diagnosis: Iatrogenic Cushing's syndrome, erythrodermic psoriasis, and hypoproteinemia. The combination of (1) adult age, (2) self-escalation to 100 g/week (twice the recommended maximum), (3) transition to erythroderma, and (4) documented HPA axis suppression within 22 months constitutes a novel pattern not previously reported.

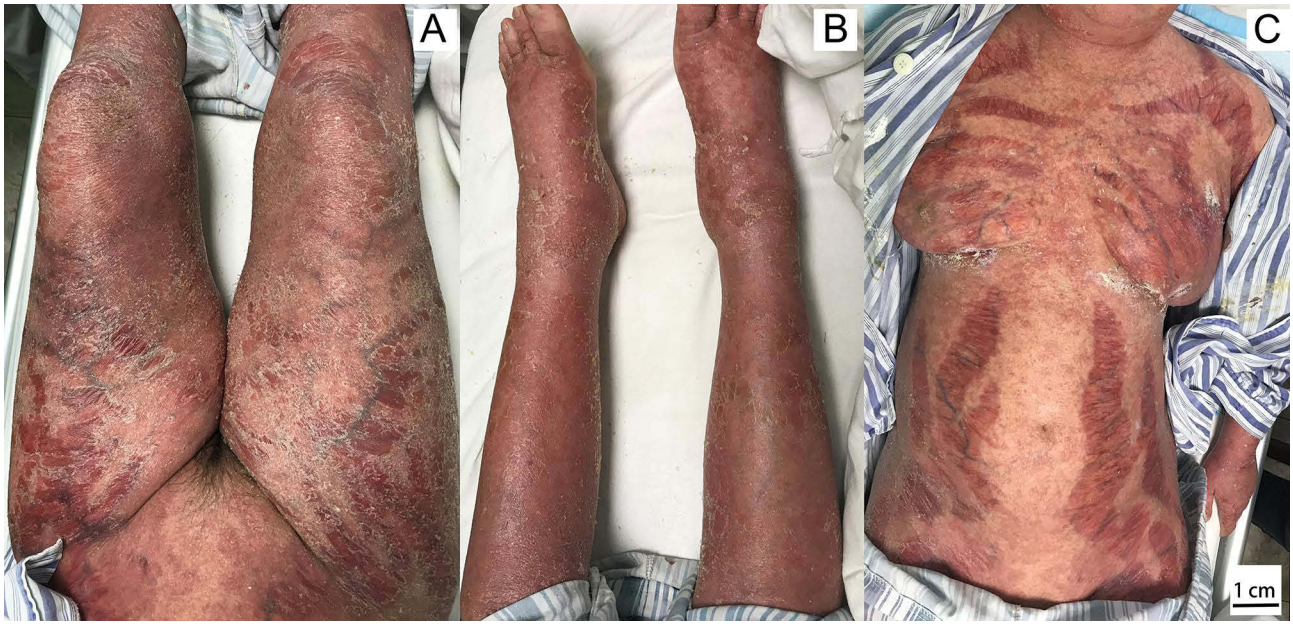

Figure 1 (**A**–**C**) Wide purple-red stripes can be seen on the patient's chest, abdomen and the outer sides of both thighs.

Discussion

Psoriasis is a chronic inflammatory dermatosis characterized by genetic predisposition and immune dysregulation. Mild to moderate psoriasis vulgaris is commonly managed with topical therapies, which may be supplemented with ultraviolet light therapy or systemic agents depending on disease severity. Standard topical treatments include glucocorticoids, vitamin D analogs, calcineurin inhibitors, and keratinocyte agents.[1] Among the severe complications of psoriasis, erythrodermic psoriasis is particularly significant, presenting with generalized erythema affecting > 90% of body surface area, frequently accompanied by systemic manifestations including fever, desquamation, and fluid-electrolyte imbalance. Psoriasis and eczema represent the most prevalent underlying causes of erythroderma, with approximately 1% to 2.5% of patients developing the erythrodermic variant.[6] Notably, excessive or inappropriate glucocorticoid administration represents a frequent precipitating factor, particularly with systemic or high potency topical formulations.[7] In this case, the patient developed erythrodermic psoriasis following prolonged intermittent application of high-potency steroids such as clobetasol propionate cream (0.05%) over a 22-month period. The absence of eczema history, coupled with erythema resolution without pruritus, supported the diagnosis of erythrodermic psoriasis rather than allergic contact dermatitis.

Topical glucocorticoids remain a cornerstone of psoriasis management. The therapeutic and adverse effects depend on multiple factors, including potency, concentration, application frequency, duration of use, treated surface area, skin barrier integrity, patient age, and concomitant use of moisturizers.[8]

Although systemic adverse effects are more frequently documented in pediatric populations due to their higher body surface area-to-weight ratio, adults remain susceptible, particularly when potent glucocorticoids are applied to extensive or compromised skin surfaces.[8,9]

Epidermal atrophy may develop as early as 3 to 14 days following initiation of topical glucocorticoid therapy. Chronic use results in stratum corneum disruption, impaired epidermal barrier function, and reduced synthesis of dermal elastic and collagen fibers, especially type I collagen. In advanced cases, atrophy may extend to subcutaneous adipose tissue, manifesting clinically as epidermal thinning, telangiectasia, atrophic striae, xerosis, desquamation, and aggravation of underlying dermatoses.[10] Percutaneous absorption of topical glucocorticoids may induce systemic effects, including HPA axis suppression, mineralocorticoid-related complications (eg, edema), and iatrogenic Cushing syndrome.[11,12]

Clobetasol propionate, a super-potent topical corticosteroid, is commonly prescribed for severe or recalcitrant psoriasis. However, its use is typically limited to no more than two to three consecutive weeks, with a maximum recommended weekly dose of 50 g.[9] Weekly doses exceeding 100 g have been associated with significant HPA axis suppression.[13]

In this case, while initial use adhered to these guidelines (clobetasol propionate at a low dose of 20–30 g/month), the presence of impaired skin barrier function and concurrent application of occlusive agents such as petroleum jelly likely facilitated enhanced percutaneous absorption. Early signs of systemic involvement, including cutaneous atrophy, emerged within eight months, yet treatment continued with escalation.

Following 14 months of high-dose application, the patient developed clinical features consistent with iatrogenic Cushing's syndrome, including moon facies, centripetal adiposity, significant weight gain (10 kg), and striae distensae (see Figure 1). These changes were attributed to glucocorticoid-induced fat redistribution, dermal collagen degradation and connective tissue injury mediated by activated macrophages and matrix metalloproteinases, contributing to the development of striae particularly in abdominal and upper limb regions.[14]

Biochemical testing confirmed adrenal suppression, as evidenced by markedly reduced plasma cortisol and adrenocorticotropic hormone levels. Hypoproteinemia, characterized by decreased serum albumin and total protein concentrations, was identified and attributed to systemic inflammation and nutritional imbalance. The diagnosis of iatrogenic Cushing's syndrome was established based on clinical presentation, biochemical findings, and treatment history. HPA axis recovery typically requires approximately 3.84 ± 2.51 months in adults, with oral corticosteroid administration based on physiological doses generally recommended between 6 to 9 months.[4]

In this case, oral methylprednisolone (8 mg daily) was administered in conjunction with low-potency topical desonide cream. Cortisol and ACTH levels normalized within four months. Although this patient's initial cumulative monthly clobetasol dose was relatively low, compromised skin barrier function and use of occlusive moisturizers significantly enhanced absorption. Consequently, systemic glucocorticoid effects developed rapidly, with concurrent exacerbation of the underlying dermatosis into erythrodermic psoriasis.

This case illustrates the catastrophic consequences of unsupervised corticosteroid escalation and absence of follow-up in chronic dermatologic care. The patient's transition to erythrodermic psoriasis likely reflects steroid-induced barrier failure and rebound inflammation, a sequence rarely captured in adult literature.

While iatrogenic Cushing's syndrome induced by topical corticosteroids have been more frequently reported in pediatric populations, similar occurrences have been documented in adults with psoriasis.[8,15,16] Joe et al described Cushing's syndrome in an 11-year-old following six months of clobetasol propionate and betamethasone dipropionate, while Abma et al reported HPA axis suppression in an adult woman following three years of intermittent topical corticosteroid use.[15,17] Compared to prior adult cases,[18,19] our patient uniquely developed erythroderma coincident with Cushingoid features, with documented monthly dose escalation rather than steady high-dose exposure.

Psoriasis management requires careful consideration of disease classification, severity, and long-term treatment strategies. Given the relapsing nature of the disease, patients may misuse corticosteroids seeking rapid symptom relief. Clinicians must remain vigilant for potential systemic complications, particularly iatrogenic Cushing's syndrome, in cases involving prolonged or high-dose topical corticosteroid use.

This report lacks histopathological confirmation of skin atrophy, relies on patient-reported dosing history, and lost dermatologic follow-up after 4 months. Future cases should include serial biopsy, photographic documentation, and prolonged endocrine/dermatologic monitoring.

Conclusions

This case demonstrates iatrogenic Cushing's syndrome in a 31-year-old patient with psoriasis following chronic, excessive use of high-potency topical glucocorticoids. The patient exhibited characteristic clinical features of glucocorticoid excess, HPA axis suppression, and hypoproteinemia. This report emphasizes that exogenous Cushing's syndrome may result not only from systemic corticosteroid administration but also from prolonged topical application, especially when factors enhance percutaneous absorption. Consequently, strict adherence to established guidelines regarding topical glucocorticoid potency, duration, and quantity is essential. Clinicians should be aware of potential systemic effects in patients undergoing long-term topical glucocorticoid therapy and ensure appropriate monitoring and patient education to mitigate iatrogenic complication risk. This case reinforces that topical corticosteroids are not benign under conditions of misuse. It advocates for (1) strict adherence to prescribing guidelines (≤50 g/week of clobetasol), (2) mandatory patient

education on systemic risks, and (3) routine screening for HPA axis suppression in high-risk scenarios. Regulatory measures limiting over-the-counter access to super-potent steroids may be warranted.

Data Sharing Statement

The datasets used and/or analyzed during the current study are available from the corresponding author upon reasonable request.

Ethics Approval and Consent to Participate

This study was conducted with approval from the Ethics Committee of Wuhan Asia General Hospital Affiliated to Wuhan University of Science and Technology. This study was conducted in accordance with the declaration of Helsinki. Written informed consent to participate was obtained from the participant.

Consent for Publication

The participant signed a document of informed consent for publication of the images.

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Disclosure

The authors declare that they have no conflicts of interest in this work.

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